treatment with IVIG was begun in the presence of hypogammaglobulinemia and recurrent bacterial infections. The treatment design consisted in 4-hr IVIG infusions at 400 mg/kg (Gammagard®, Baxter, Glendale, CA) every 3 weeks. While no side effects were noted during the first infusion, the patient described a feeling of coldness beginning a few hours after hospital discharge and lasing for 2 days. After the second IVIG infusion, the patient reported the same side effect. His body temperature was 35°C, a lowering of 1.5°C compared to the preinfusion value. This observation was particularly obvious after the first three IVIG administrations, and then the condition subsided by itself. The underlying mechanisms of this reaction remain largely speculative. The B-CLL of the patient was stable, and he did not present any endocrinopathy other than a well-balanced insulin-dependent diabetes. The chronology of symptoms makes the hypothesis of a direct cooling effect of insufficiently heated IVIG batches very unlikely. Hypothermia is a drug reaction far less frequent than fever, especially ouside of an obvious toxic context. A direct effect of aggregated immunoglobulins and/or of circulating antibody-antigen complexes at the hypothalamic thermoregulatory center can be postulated. It is noteworthy that in our patient the dosage of various complement fractions was always decreased with C3, C4, and C1Q inhibitor levels below 50% of normal values, as we have reported in other B-CLL patients [1], reflecting a chronic activation of the complement system. Interactions between IVIG and an altered complement system could have induced alterations in the kininogen pathway, the central, hypothalamic, and vasomotor effects of which are well-documented in rodents and hypothesized in humans [2]. This particular feature of CLL could be relevant for the reported reaction.

Hypothermia can be added to the few reported neurological complications occurring after IVIG infusions, e.g., aseptic meningitis [3,4] and recurrent migraine [5].

Caroline Duhem
Fernand Ries
Mario Dicato

Department of Hematology-Oncology, Centre Hospitalier de Luxembourg, Luxembourg, Luxembourg

References

1. Dicato M, Schmit JC, Mahon G, Ries F: Chronic activation of complement in chronic lymphocytic leukemia. Blood [Suppl] 78:464, 1991.
2. Richoux JP, Gelly JL, Bouhnik J, Baussant T, Albenc-Gelas F, Grignon F, Corvol P: The kallikrein-kinin system in the rat hypothalamus. Immunohistochemical localization of high molecular weight kinoinogen and T-kininogen în different neuronal systems. Histochemistry 96:229–243, 1991.
3. Kato E, Shindo S, Eto, Y: Administration of immune globulins associated with aseptic meningitis. JAMA 259:3269–3271, 1988.
4. Casteels-Van Daele M, Wijndaele L, Hunninck K, Gillis P: Intravenous immune globulin and acute aseptic meningitis. N Engl J Med 323:614–615, 1990.
5. Constantinescu C, Chang A, McCluskey L: Recurrent migraine and intravenous immune globulin therapy. N Engl J Med 329:583–584, 1993.

Multiple Spontaneous Vascular Infarcts In Sickle-Cell Trait: A Case Report

To the Editor: Vascular occlusive crisis is the most common complication of sickle-cell disease. This phenomenon, however, is exceptional in sickle-cell trait. We report on a case of multiple vascular infarcts in a patient with sickle-cell trait.

A 65-year-old North African woman was admitted for facial neuralgias and left upper quadrant abdominal pain. Clinical exam was unremarkable. No biological abnormality, including arterial oxygen tension, was seen, except for hemoglobin electrophoresis which demonstrated Hb A1 57.7%, HbS 40.3%, and HbA2 2.2%. Itano test was positive. Hemogram was as follows: Hb 120 g/l, platelets 375 × 10^9/l, leucocytes (WBC) 9.7 × 10^9/l with a normal differential count. During hospitalization, abdominal pain increased. An abdominal ultrasound was performed and did not reveal any abnormality. Despite this, abdominal pain worsened. Fifteen days after, a computerized tomographic (CT) scan revealed a marked splenomegaly with a hypodense mass (9 × 6 cm) consistent with a splenic infarction. Splenectomy confirmed the diagnosis. During the surgical procedure, arterial oxygen tension was monitored and did not fall under 95%. The patient was then referred to us. On admission, the patient was confused, with no other clinical abnormality. A cerebral CT scan showed two recent infarcts. On room-air arterial blood gas studies, oxygen tension was 73 mg Hg. Then, a pulmonary scintigraphy was performed, which showed multiple pulmonary infarctions. Anticoagulant therapy was started. With a follow-up of 2 years, the patient is still doing well.

Several investigations were performed to understand these abnormalities. There was no arterial hypoxemia before splenic infarction, nor acidosis. Electrocardiogram, and transthoracic and transesophageal cardiac ultrasonography, were normal. Doppler ultrasound did not reveal any deep venous thrombosis. Routine tests exploring hemostasis were normal. There was no deficit in antithrombin III, and proteins S and C, and no resistance to activated protein C. Antiphospholipid antibodies were not found. Fibrinolysis, platelet functions, erythrocyte density, and dosage of 2–3 diphosphoglycerate (DPG) were unremarkable. Analysis of the β-globin gene did not show any abnormality other than a mutation on codon 6 on one allele.

Sickle-cell trait is usually asymptomatic. Splenic infarction has been described in patients who were flying in an unpressurized aircraft or who ascended to high altitudes. Apart from these extreme conditions, only exceptional cases of splenic infarction have been reported. In our patient, suffering from multiple severe thombotic processes without predisposing factors, no abnormality other than sickle-cell trait was found. Our observation, with those of others, must alert physicians to the possibility, even in the absence of hypoxemia, of serious thrombotic events in these patients.

Philippe Genet
Marc Pulik
François Lionnet
Charlotte Petitdidier
Tahar Touahri

Department of Hematology, Victor Dupouy Hospital, Argenteuil, France

Rerences

1. Lane PA, Githens JH: Splenic syndrome at mountain altitudes in sickle cell trait. JAMA 253:2251–2254, 1985.
2. Gitlin SD, Thompson GB: Non-altitude-related splenic infarction in a patient with sickle cell trait. Am J Med 87:697–698, 1989.
3. King DT, Lindstrom RR, State D, Hirose FM, Schwartz A: Unusual cause of acute abdomen: Sickle cell trait and nonhypoxic splenic infarction. JAMA 238:2173–2174, 1977.
4. Magnusson TR, Hunter SW, Bonnabeau RC: Multiple vascular infarction: A manifestation of sickle cell trait in the absence of hypoxia. Minn Med 63:381–383, 1980.

High-Dose Biscoclaurine Alkaloids Together With Prednisolone Raise Platelet Counts in Chronic Idiopathic Thrombocytopenic Purpura

To the Editor: Chronic idiopathic thrombocytopenic purpura (ITP) is a syndrome characterized by persistent thrombocytopenia caused by a circulating antiplatelet factor which results in platelet destruction in the reticuloendothelial system. Generally, ITP is treated with glucocorticoids, androgens, vinca alkaloids, cyclophosphamide, azathioprine, anti-Rhesus globulin, high-dose immunoglobulins, colchicine, ascorbate, and splenec-